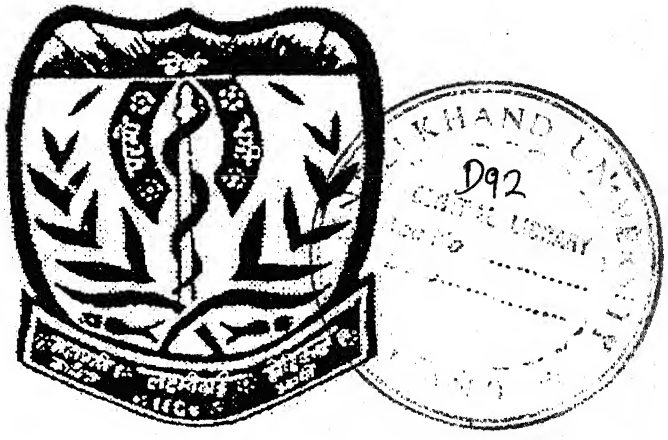
DEPARTMENT OF MEDICINE

ECHOCARDIOGRAPHIC EVALUATION OF CARDIAC ABNORMALITIES IN CHRONIC RENAL FAILURE PATIENTS

THESIS

FOR

DOCTOR OF MEDICINE
(INTERNAL MEDICINE)



BUNDELKHAND UNIVERSITY
JHANSI (U.P.)

2005

VIJAY KUMAR

Dedicated

To

My Parents

CERTIFICATE

This is to certify that the work entitled ***"Echocardiographic evaluation of cardiac abnormalities in chronic renal failure patients"*** which is being submitted as a thesis for M.D. (Medicine) Examination 2005 of Bundelkhand University, Jhansi, has been carried out by ***Dr. Vijay Kumar*** in the Department of Medicine, M.L.B. Medical College, Jhansi.

This method described was undertaken by the candidate himself and the observations recorded have been periodically checked up. He has put in the necessary stay in the Department as per University regulations, and has fulfilled the conditions required for the submission of thesis according to University regulations.

Dated: 19/10/04

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M.D., MNAMS
Assoc. Professor & Head,
Department of Medicine,
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Dated: 19/10/04

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Dated: 19/10/04

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(Co-Guide)

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Dated: 19/10/04

Sethi

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(Co-Guide)

Acknowledgement

It would take pages, to acknowledgement everyone who in sometime who in someway or the other helped me along my path reaching this goal, but certain individuals need special thanks for their valuable help.

First and foremost, with all my heart I would like to express my deepest gratitude and privilege for having been associated with my ***Guide Dr Praveen Kumar Jain*** *MD. DM, Professor of Cardiology, Department of Medicine Maharani Laxmi Bai Medical College, Jhansi for his enlightened guidance which steered me towards the harbour of success. I am greatly thankful for the ceaseless attention he has paid to my problems, always sparing a few moments for me even amidst the busiest of schedules without which this work would not have seen the light of day.*

"The habits of punctuality, order, diligence, determination and concentration are the key to success ". That is what has been inculcated into us by our honorable ***Head of Department Dr. P.K. Jain,*** *MD, MNAMS, Professor, Department of Medicine, M.L.B. Medical College. Jhansi.*

I am also greatly thankful to my ***Co-Guide Dr N.S. Senger*** *MD, DM, Assistant Professor in Nephrology, Department of Medicine M.L.B. Medical College, Jhansi. His sense of precision, inflinching tenacity, passion for reason, compassion towards patients, knowledge and experience was a constant source of inspiration to me. It was under his able guidance I read, learned and ventured to write. I take this opportunity to acknowledge most humbly with a deep sense of gratitude and indebtedness to my* ***Co-Guide Dr R.S. Sethi****, MD, D.Ch,*

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I shall faith all my thanks if do not convey all my thanks and respect to all the people who took part in this work and helped me to complete this study and reach conclusion.

Nothing in this work can reply the efforts, sacrifices and support of my parents. Their constant support and morale boosters always pulled me out of the dark.

Vijay Kumar

Dated: 19/10/04

(Dr. Vijay Kumar)

Contents

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Introduction

Introduction

Chronic or irreversible renal failure is a constellation of signs and symptoms, which result from progressive and irreversible renal damage. It leads to various endocrinal, metabolic and electrolyte disturbances and affects virtually every organ system in the body. The successful treatment of end stage renal disease (ESRD) by dialysis and renal transplantation is considered one of the major advances of modern medicine.

Cardiovascular diseases is the leading cause of death in the patients with ESRD (USRDS, 1993). According to US Renal Data Systems (USRDS) (1993) cardiovascular diseases account for nearly 40% of death among patients with chronic renal failure (CRF). Infact during the last 3 decades cardiovascular mortality in the general population in the United States has decreased by 20% to 30% [Stern MP (1979), Levy RI (1982)]. In contrast during the same time cardiovascular mortality has increased to about 40% in patients with chronic renal disorder.

In an autopsy review of dialysis patients, 31% were noted to have pericardial fibrosis or pericarditis, ventricular dilatation was

observed in 47%, left ventricular hypertrophy (LVH) was noted in 66% right ventricular hypertrophy was seen in 33% and biventricular hypertrophy was seen in 33% (Ansari et al 1993).

Two-D-Echo is a non invasive technique, which is easily applied and provides information about the size and function of the ventricles and wall thickness and about the function of the valves.

Echo has proved to be of great help in the assessment of symptomatic patients with C.R.F not only in diagnosing the presence and extent of pericardial effusion and thickening, but also in detecting impaired myocardial contractility, calcification and vegetations of bacterial endocarditis.

L.V.H. in chronic renal failure: L.V.H. is common in chronic renal failure and is found in approximately 70% of dialysis patients.

The pattern of L.V.H. may be symmetric, with thickening of both the septum and posterior wall but normal cavity dimensions L.V.H. may also follow an eccentric pattern where there is accompanying dilatation of the left ventricular cavity.

Increased left ventricular mass is accompanied by increased size of individual cardiac myocytes. Echo is undoubtedly the method of choice to assess left ventricular geometry.

C.H.F in chronic renal failure patients

The occurrence of C.H.F and systolic left ventricular dysfunction in uraemic patients is clearly related to history of hypertension and is usually do a combination of I.H.D and hypertensive injury.

In a cross sectional study C.H.F, as defined by persistent or recurrent heart failure when patients were considered to be at dry weight was found in 10% non-diabetic dialysis patients.

Valvular Heart disease in chronic renal failure

Following the report of Linder et al (1974), it was hypothesized that atherogenesis is accelerated in renal failure. Calcific A.S M.S. metastatic myocardial calcifications are common.

A high prevalence of aortic valve calcification (55%) and A.S. (13%) has been reported in recent studies using 2 D echo. The

figures for mitral valve calcification, M.R. and M.S. were 40%, 4% and 5% respectively.

Uraemic Pericarditis and Pericardial effusion in chronic renal failure

Pericardial effusion is also found in C.R.F. patients of pericarditis suspected, echo should be performed to recognize pericardial effusion.

Endocarditis

Uraemic patients are at increased risk of developing endocarditis. Echo including transoesophageal echo is recommended in patients with suspected endocarditis valvular vegetations may be detected, but this is not a constant finding. Valvular destruction may be rapid. Echo is particularly important for detecting incipient valvular dysfunction.

Review Of Literature

Review of literature

Cardiac problems in patients with renal insufficiency

Epidemiology

Cardiovascular accidents are the most frequent cause of death dialysis patients. According to the US Renal Data System (1995) cardiac causes accounted for 41.3 percent and carebrovascular disease for 5.6 percent of all deaths. The rates, but not the proportions were greater in male and diabetic patients. These findings are in good agreement with European data (European Dialysis and Transplantation Association Registry 1995). Increased attention to health promotion and preventive strategies, particularly in the United States has resulted in an absolute decline of mortality from ischaemic heart disease in the general population. The same is not true for endstage renal disease. The magnitude of the risk in the dialysed patient is evident from comparison with other high-risk groups

When mortality from cardiovascular causes in patients with endstage renal failure in northern Europe is compared with that in

similar patients in southern Europe, it is striking that mortality rates from stroke, heart failure and sudden death are relatively similar whereas the incidence of death from ischaemic heart disease is three to four times greater in north European patients. Age, gender and country specific rates of death from myocardial infarction relative to the population are always increased by a factor of approximately 18.

Factors affecting cardiac function and structure in renal failure

Left ventricular hypertrophy is the most frequent cardiac abnormality in patients with endstage renal disease, with a prevalence ranging from 30 to 80 per cent (Ikram et al. 1983; Bernardi et al. 1985; Kramer et al. 1986a; Kramer et al. 1986b; Parfrey et al. 1990a; Ruffman et al. 1990). Invasive studies of unselected dialysis populations have shown that cardiac dilatation is rare in the absence of ischaemic heart disease (Wizemann and Kramer 1988) and tends to be associated with anemia, arteriovenous fistula, flow, and poor control of volume overload (Drueke et al. 1977; London et al. 1987a; London et al. 1989a).

Systolic function is usually normal, or even increased, but evidence of diastolic malfunction, including reduced compliance and

abnormal passive filling of the left ventricle, can be found even in asymptomatic patients. Diastolic filling problems are strongly associated with left ventricular hypertrophy of the concentric or asymmetrical (septal) type.

Some important factors influencing left ventricular function and mass are listed below. Some of these factors will he discussed below

Hypertension

In non-renal patients hypertension is closely related to left ventricular hypertrophy, but this relationship is less marked in patients with renal failure (Huting et at. 1988; Amann et al. 1996). Left ventricular hypertrophy develops in uraemic animals despite normalization of blood pressure using angiotensin-converting enzyme inhibitors, α and β-blockers, or diuretics (Rambausek et al. 1985). In humans left ventricular hypertrophy progresses with time on dialysis even when patients are normotensive (Huting et al. 1988; Parfrey et al, 1990a). In the study by Parfrey et al. (1990a) 71 percent of non-diabetic dialysis patients without dilated cardiomyopathy had left ventricular hypertrophy, and in the majority of patients this progressed over a period of 3 to 4 years; Progression could not be

predicted on the basis of blood pressure, hyperparathyroidism, or anemia, which suggests that other factors are involved in addition to hypertension.

Haemodynamic factors

Hypertension

Fluid retention

Renal anaemia

Arteriovenous fistula

Acquired valvular disease

Constrictive pericarditis

Non-haemodynamic factors

Ischaemic heart disease

Diabetic cardiomyopathy

Autonomic dysfunction

Excess PTH

Aluminum overload

Hypocalcaemia

Myocardial calcification (calcium-phosphorus product)

Metabolic acidosis

Iron overload

B_2- M-amyloidosis

Thiamine deficiency, carnitine deficiency (?)

Drug toxicity

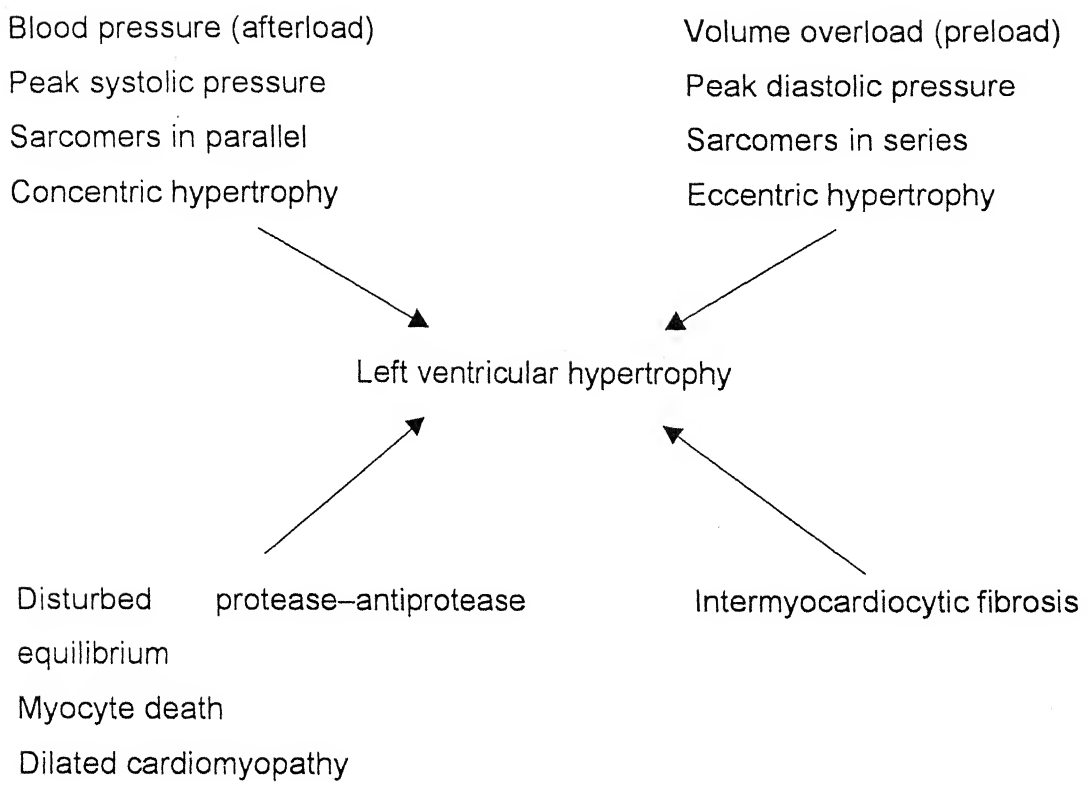
Left ventricular hypertrophy and structural abnormalities of the heart

Left ventricular hypertrophy is present in 60 to 80 per cent of patients who are admitted for treatment of endstage renal failure (Harnett et al. 1988; Foley et al. 1992; Parfrey et al. 1996), but it develops early in the course of renal failure (Fabbian et al, 1994; Stefanski et al. 1996). Although there is some degree of regression after successful renal transplantation, it remains common even when patients are normotensive and is closely related to 24-h systolic blood pressure (Lipkin et al. 1993).

The pattern of left ventricular hypertrophy may be symmetric, with thickening of both the septum and the posterior wall but normal cavity dimensions. This form is closely associated with hypertension. Left ventricular hypertrophy may also follow an eccentric pattern, where there is accompanying dilatation of the left ventricular cavity

(London et al. 1989a). Some authors have also suggested that asymmetric left ventricular hypertrophy with primarily septum thickening (S/P> 1.4) is common in dialysis patients, although this is still in dispute (Bernardi et al. 1985; Heng et al. 1985; Huting et al. 1988). As discussed in detail above, although blood pressure undoubtedly plays a major role in the genesis of left ventricular hypertrophy, other factors must also he involved. This is suggested by animal experiments (Rambausek et al, 1989) and clinical observations (Huting et al, 1988, Parfrey et al, 1990a).

Increased left ventricular mass is accompanied by increased size of individual cardiac myocytes, as seen in other settings of cardiac hypertrophy. and this is associated with increased cytosolic calcium (McMohan et al, 1995). Left ventricular hypertrophy partially reverses after transplantation (Ikaheimo et al,1982), but Lipkin et al (1993) noted increased mass despite normal casual blood pressure. Left ventricular mass was closely related to ambulatory blood pressure.



The increase in cardiomyocyte mass in left ventricular hypertrophy is accompanied by additional changes of heart structure, which may impact, on function. These comprise interstitial myocardial fibrosis and myocardial calcification.

Cardiac fibrosis in uraemic patients has been known for many years (Rossle 1943; Langendorf and Pirani 1947). It is common in patients on dialysis (Mall et al. 1990) and can be reproduced in uraemic animals (Mall et al, 1988). It is obvious that there is considerable expansion of the interstitium with deposition of collagen

fibres at the expense of capillaries. Ultrastructural analysis shows activation of interstitial cells. Activation selectively concerns interstitial, but not endothelial cells. Interstitial cell activation is accompanied by increased expression of cytokines, particularly transforming growth factor-β. In subtotally nephrectomized rats interstitial fibrosis is not consistently prevented by antihypertensive treatment (Tornig et al. 1996) but is significantly reduced by parathyroidectomy (Amann et al, 1994).

Logistic regression was used to relate intermyocardiocytic fibrosis to uraemia per ce independent of hypertension, diabetes mellitus, anaemia, heart weight, and type of dialysis. The lesions were symmetrically more severe in the right than in the left ventricle, arguing against a major role of haemodynamic overload.

A further structural change observed in the hearts of uraemic patients is metastatic calcification. It is most closely related to persistent elevation of the calcium-phosphate product (Maher et al, 1987). Rostand et al (1994), employing energy subtraction analysis, showed the subclinical myocardial calcification was common in dialysis patients. It was associated with left ventricular dysfunction with a background of previous parathyroid disease.

The structural changes in the heart, i.e. left ventricular hypertrophy intercardiomyocytic fibrosis and metastatic myocardial calcification have major implications for cardiac function. They result in impairment of left ventricular compliance, which is demonstrable by Doppler echocardiography. The reduction of left ventricular compliance has several consequences. It impairs early left ventricular filling thus decreasing the relative contribution of passive protodiastolic filling to active atrial contraction. Reduction of compliance also has an adverse effect on the relationship between volume and pressure, as a consequence hypervoleamia will be less well tolerated because the segmented intracardiac volume will cause a more marked increase of left ventricular end-diastolic pressure, thus increasing the risk of pulmonary oedema. Conversely, a marked reduction in intravascular volume and left ventricular end-diastolic pressure, resulting in diminished cardiac output. This may explain why intradialytic hypotension is much more frequent in patients with left ventricular hypertrophy than in those without (Ritz et al, 1987; Wizermann et al, 1993b).

In addition to affecting myocardial compliance, left ventricular hypertrophy also adversely affects coronary reserve, i.e. the ability of

coronary arteries to dilate in order to meet increased myocardial demand. This abnormality predisposes the heart of the uraemic patient to ischemia intolerance in situations of increased oxygen demand. Finally, it is likely that disruption of normal cardiac anatomy by fibrotic strands and calcified deposits predisposes the uraemic heart to increased polythymogenesis, particularly re-entrant arrhythmia, since interposition of high resistance areas will set the stage for re-entry pathways.

Against the background it is not surprising that left ventricular hypertrophy is an independent predictor of cardiac death in dialysis patients when assessed by multivariate analysis (Silberberg et al, 1989a).

Traditional methods of diagnosing left ventricular hypertrophy are inappropriate and misleading in dialysis patients. A powerful apex beat, a third and fourth heart sound, and an increased cardiothoracic ratio on chest radiography may be caused by hypervolaemia. The ECG lacks sensitivity. Echocardiography is undoubtedly the method of choice to assess left ventricular geometry. It is important that a standardized approach is used, with initial location of the echo beam through the mitral valve apparatus, and subsequent identification and

measurements of the septum and the posterior wall by M-mode recording. The usual procedure is then to derive left ventricular mass by the Penn convention (Devereus and Reichek, 1977). The results are strongly dependent on observer experience.

Congestive heart failure

The difficulties of distinguishing congestive heart failure from circulatory congestion have been discussed above. In a cross-sectional study congestive heart failure, as defined by persistent or recurrent heart failure when patients were considered to be at dry weight, was found in 10 per cent of non-diabetic dialysis- patients (Parfrey et al, 1990b). Ancillary non-specific findings are a history of dyspnoea or peripheral oedema, cardiomegaly, increased jugular venous pressure, basal crepitations, pulmonary venous hypertension, or interstitial oedema on chest radiography. A third of the patients had developed their congestive heart failure before reaching endstage renal failure, 53 percent of these patients had dilated cardiomyopathy and 47 percent had hypertrophic hyperkinetic disease. Dilated cardiomyopathy affecting systolic pumping function, as characterized by a, low ejection fraction and increased end-systolic and end

diastolic diameters, is not particularly common In dialysis patient. The most common cause of congestive heart failure is ischaemic heart disease; but other factors for example diabetic cardiomyopathy, myocardial calcification, iron overload, and thiamine deficiency, may also be involved. Congestive heart failure carries a poor prognosis; actuarial 2-years survival is no more than 33 per cent compared with 80 percent in patients without congestive heart failure (Parfrey et al, 1990b). A remarkable reversal of left ventricular dysfunction was noted after renal transplantation in some patients (Burt et al, 1989), but this is not a consistent outcome.

Valvular heart disease

The risk of accelerated calcific valvular heart disease in the dialysis patient was not adequately appreciated in the past (Raine 1994). Calcific aortic stenosis, mitral valve calcification,, and (mostly subclinical) metastatic myocardial calcification are common. A high prevalence of aortic valve calcification (55 percent) and aortic stenosis (13 percent) has been reported in recent studies assessing two-dimensional echocardiography (Raine 1994). The figures for mitral valve calcification, mitral regurgitation and mitral stenosis were

40 percent, 11 percent, and 5 percent respectively. A recent Italian survey (Mazzaferro et al, 1993) found that mitral valve calcification was completely absent in non-anemic subjects aged less than 60 years, while it was found in 37 percent of dialysis patients. Since age is a risk factor for the development of valvular calcification, uraemia can be considered as a state of accelerated ageing. Risk predictors comprise of duration of dialysis, hyperphosphataemia, hypercalcaemia, and elevated PTH (Maher et al, 1987). In some patients calcific aortic stenosis progresses with extreme rapidity (Maher and Curtis 1985), particularly in the presence of severe hyperparathyroidism. The severity of aortic stenosis can easily be underestimated by clinical examination. It should also be remembered that calcific valvular disease is frequently associated with conduction defects (Terman et al. 1971). The patient should be managed to maintain serum phosphate less than 1.9 mmol/L. Overt hyperparathyroidism should be corrected. If valve replacement is eventually indicated, the use of a prosthetic valve is appropriate to minimize the risk of calcification since bioprostheses carry an excessive risk of calcification in uraemic patients.

Uraemic pericarditis and pericardial effusion

In the past uraemic pericarditis was regarded as one of the final events before death in uraemic coma ensued. However, dialysis has clinically improved the prognosis of uraemic pericarditis. Nevertheless it continues to be an important clinical problem. A certain proportion of patients on maintenance dialysis suffer single or recurrent episodes of pericarditis. The major causes are intercurrent viral infection or underdialysis (due to insufficient fistula flow, duration of dialysis sessions, or efficacy of dialysis).

Diagnosis

The patient with uraemic pericarditis often, but not invariably, presents with pericardial pain, which may resemble angina pectoris. Cardiac arrhythmia, particularly atrial fibrillation, may also he present. Dyspnoea and other symptoms of congestion occur when large effusions and / or pericardial constriction develops. On clinical examination a systolic - diastolic-pericardial friction rub can he heard which typically comprises three components. This important physical sign often disappears with the development of larger effusions. They, cause inspiratory distension of jugular veins instead of inspiratory

collapse (Kussmaul's.sign) and may lead to pulsus paradoxus (decrease of systolic pressure upon inspiration). Hypotension in combination with narrowed pulse pressure is an alarming sign, suggesting imminent cardiac, tamponade. However, not all uraemic patients with pericarditis are symptomatic. If pericarditis is suspected, echocardiography should be performed to recognize .pericardial effusion. Repeated echocardiography is mandatory to monitor the response of effusion to therapy. Less precise information is provided by chest radiographs and electrocardiography. The absence of pulmonary congestion, despite enlargement of the heart, distinguishes pericardial effusion from congestive heart failure, but extreme effusions may also compromise left ventricular filling and occasionally lead to pulmonary congestion. The electrocardiogram is not reliable in the diagnosis of uraemic pericarditis. However, a low-voltage QRS segment is commonly found in patients with marked pericardial effusion, and beat-to-beat variation of the heart axis (swinging heart) may occur. Long-term sequelae of pericardial effusion, particularly, of the haemorrhage type, are pericardial constriction and calcification.

Aims

&

Objectives

Aims and Objectives

Echocardiographic evaluation of cardiac abnormalities like Left Ventricular Hypertrophy, cardiac calcifications, pericarditis Congestive Heart Failure infective endocarditis in chronic renal failure patients.

Material & Methods

Material and Methods

60 patients of Chronic Renal Failure were taken from Medical O.P.D. , Nephrology clinic and wards.

Patient's name. Age, sex, occupational history, history of alcohol intake, smoking, tobacco / gutka chewing and other dietary habits, history of diabetes, obesity, Myocardial Infarction, weight loss, hematuria. Drug history NSAIDS and other relevant history was taken along with the family history of hypertension, C.A.D. diabetes, obesity etc were also taken.

Inclusion criteria : Only those patients were taken whose blood urea and serum creatinine were above normal with reference to age and sex for more than 3 months.

Those patients have not been included who have primary cardiac abnormalities which were not because of chronic renal failures.

For diabetic amyloidosis and renal stone disease chronic renal failure patients kidney size were normal or increased but they were fulfilling the criteria of chronic renal failure on the basis of increased

serum creatinine for more than 3 months duration were also having other findings like urine (R and M), retinopathy and anemia.

Patient's complete examination was done followed by routine investigations, e.g. Blood Sugar, Blood urea, S.Creatinine, C.B.C, urine (routine and microscopy), X-ray chest PA-view, 12 lead E.C.G. and fasting lipid profile. 60 patients of CRF were taken which were having an age group between 8 – 55 years, out of which 40 were males and 20 were females. These patients were randomized into three groups i.e. mild chronic renal failure (Group A), moderate chronic renal failure (Group B) and severe chronic renal failure (Group C) according to their increased serum creatinine levels for more than 3 months and other parameters supporting CRF. Mild chronic renal failure patients having serum creatinine in the range of 1.5 – 3 mg/dl, moderate chronic renal failure serum creatinine in the range of 3 – 6 mg/dl and severe chronic renal failure patients having serum creatinine more than 6 mg/dl, and they have been evaluated by method and working proforma given as follows:

The left ventricular dysfunction will be asserted in following headings:

1. L.V.H
2. Diastolic dysfunction
3. Systolic dysfunction

L.V.H. : There are two methods of calculating L.V. mass from 2-D echocardiography-

a. Area length method

b. Truncated ellipsoid method

For both methods short axis view of left ventricle at papillary muscle level and apical four or two chamber view at end diastole are required. Myocardial mass is equal to product of volume and specific gravity of myocardium (1.04 gm/ml). Built in software in ultrasound unit can make both methods available so that mass is automatically calculated once all variables are fed. LV mass can also be estimated from 2-D guided, M mode measurement of LV dimension and wall thickness at papillary muscle level without measuring left ventricular major axis. Left ventricular mass is reliably obtained from left

ventricular short axis dimension and simple geometric cube formula. The following equations provide accurate determination of LV mass according to Devereux and associated.

Left ventricular mass (gms) = 1.04 [(LVID + PWT + IVST)3 – LVID3] X 0.8 + 0.6

Where:-

- 1.04 specific gravity of myocardium
- 0.8- correction factor
- LVID-Left ventricle internal dimension
- PWT-Posterior wall thickness
- IVST-Interventricular septal thickness measured at end diastolic.

Diastolic dysfunction

Based on Doppler velocity pattern, diastolic dysfunction is divided into three category.

a. Relaxation abnormalities

b. Restrictive physiology

c. pseudo normalization

a. Relaxation abnormalities: - Abnormal myocardial relaxation is characterized by a constellation of following abnormalities.

1. Prolonged IVRT (Isovolumic relaxation time)> 110 msec.

2. Low F2 velocity (early filling velocity) and high A velocity (A velocity = late filling velocity).

3. Revised E/A ratio (<1.0)

4. Prolonged declaration time (DT) > 240msec.

b. Restrictive physiology: Is characterized by following diastolic parameters:

1. Shortened IVRT (<60msec)

2. High E & velocity and low A velocity

3. Increased T2 ratio> 2.

4. Shortened deceleration time (< 150msec)

Systolic dysfunction

To evaluate systolic function two parameters are used:

1. Fractional shortening or ejection fraction.

2. Cardiac output

1.Farctional shortening is a percent change in left ventricle cavity dimension with systolic contraction and can be calculated from the following equation.

Fractional shortening = (LVED-LVES)/LVED x 100%

Where LVES-LV end systolic dimension

LVED - LV end diastolic dimension

2. Ejection fraction:

Represents stroke volume as a percent of end diastolic left ventricular volume-

EF= (EDV-ESV)/EDV x 100%

Where EDV-End diastolic volume of LV

ESV-End diastolic volume of LV

Quinines and co-authors proposed a simplified method for determination of ejection fraction by measuring left ventricle dimension.

$IF = (\%OD^2) + [(1-\%OD^2) (\%DL)$

Whereas- $\%OD^2$ [(LVED-LVES0/LVED] x 100%

EF- $(\%OD^2) + [(1 +\%OD^2)][\%OD]$

Where $\%OD^2$=LVED-LVES/LVED x100%

Fractional shortening of the square of minor axis %OD= fraction, shortening of long waits mainly related to apical contraception where

15% is normal

5%= hyperemic apex -5% -madly dyslirnescafea

0%- kinetic codex -10%- apica

Evaluation of valvular stenosis

A stenotic valve is generally thickened & calcified. & its opening is restricted which can be visualized by 2.D echo blood flow velocity across a stenotic valve increases as the orifice area becomes smaller. From Doppler velocity (V) transvalvular pressure gradients can be derived based on the modified Bernoulli equation.

$$\text{Pressure gradient} = 4 \times V^2$$

The continuity equation, derived from the same basic hydraulic formula on which the Gorlin formula is based, can reliably estimate valve area by calculating stroke volume form another cardiac orifice.

Pressure half time (P.H.T.) , which is the time interval required for the peak pressure gradient to reach one half of its initial value, is another useful Doppler parameter to asses the severity of M.S. A.S.

The normal aortic valve area is 3-4 cm^2 & normal opening excursion generally produces 2.0 cm of leaflet separation.

Doppler Echo:- The hemodynamic severity of A.S. determined by Doppler is based on peak aortic velocity , mean pressure gradient, aortic valve area.

L.V.O.T. aortic valve time velocity integral

Method

Step –1	Obtain the maximum aortic jet velocity by systemic search utilizing windows
Step –2	Calculate the mean aortic gradient and TV_1 by tracing the maximum velocity jet
Step –3	Determine stroke volume and cardiac output from LVOT diameter and velocity
Step –4	Calculate aortic valve area using the continuity equation

Stroke volume across the aortic valve needs to be calculated to determine aortic valve area. This is done by measuring the L.V.O.T diameter & flow velocity.

(A) Measurement of L.V.O.T. diameter (D): from the systolic freeze frame of the parasternal long axis view.

The distance from where the anterior aortic cusp meets the ventricular septum to the point where the posterior cusp meets the anterior mitral leaflet.

(B) Measurement of L.V.O.T. velocity from the apical.

Long axis view

The pulsed wave sample volume is located 3-5mm proximal to the aortic value.

(C) Determination of L.V.O.T. TV1 TV1 is determined by tracing the velocity envelope & is equal to the sum of individual velocities of the Doppler spectrum.

Although both spectra show a similar peak velocity (.8m/sec) the TV_1 is markedly different due to the difference in ejection time (E.T.)

L.V.O.T. == L.V.O.T. area x L.V.O.T, TV_1

$= (D/2)^2 \times n \times$ L.V.O.T. TV_1

$= D^2 \times .785 \times$ LV.O.T.TV_1

D = Diameter of L.V.O.T.

The Continuity equation :- States that the flow across the L.V.O.T. is the same as the flow across the aortic valve therefore

L.V.O.T. flow = A.V. flow

L.V.O.T. $D^2 .785 \times$ L.V.O.T. $TV_1 =$ AVA x $AVTV_1$

AVA = L.V.O.T. $D^2 \times .785 \times$ L.V.O.T. $TV_1 / AVTV_1$

Where D = diameter, * AVA = aortic value area

Since flow duration across L.V.O.T. & the aortic valve is same , the TV_1 ratio is similar to their peak velocity (V) ratio.

Therefore the contumely equation can be simplified to

$$AVA = D^2 \times .785 \times LVOTV / AVV$$

It should be noted that TV_1 or peak velocity ratio is inversely proportional to the area ratio of the L.V.O.T. & aortic valve.

Mitral Stenosis

2.0. 8 M- mode Echo:- The mitral valve area can be measured by planimetry from the parasternal short axis view .

Doppler & color flow imaging :- it estimates severity of M.S. These include mitral pressure gradients, P.H.T, mitral valve area, pulmonary artery pressure , & associated regurgitation .

Estimation of mitral stenosis severity:-

Step –1	Determine the pressure gradient by obtaining the maximum velocity using continuous wave Doppler from the apical and par apical positions
Step –2	Obtain the mean gradient and TV_1
Step –3	Calculate the mitral valve area (M.V.A.) M.V.A. = L.V.O.T.D^2 x 0.785 x L.V.O.T.TV_1 / $MVTV_1$
Step –4	Determine the pulmonary artery pressure by utilizing the tricuspid regurgitation velocity

Pericardial effusion

When the potential pericardial space is filled with fluid or blood it is detected as an Echo free space. When the amount of effusion is > 25 ml Echo free space persists throughout Cardiac cycle. A Smaller amount of pericardial effusion may be detected as a posterior echo free space present only during systolic phase. When fluid small - only seen in diastole. When fluid is mild - seen in diastole & systole. When fluid is moderate -< 1 cm

When severe pericardial effusion -> 1 cm

Evaluation of Valvular Regurgitation

2D/M- mode echocardiography is useful in the assessment of valvular regurgitation by demonstrating the structural substrate for regurgitation (i.e. mitral valve prolapse, endocarditis, bicuspid aortic valve, annulus dilatation, carcinoid, etc), measuring LV size and global systolic function, and occasionally revealing the hemodynamic impact of regurgitation. However, the severity of valvular regurgitation cannot be assessed reliably by 2D echocardiography alone.

Aortic Regurgitation

2D/M-mode Echocardiography

The etiology of AR varies widely and includes congenitally abnormal valve, dilated aortic root, Marfan's syndrome, endocarditis, aortic dissection, prosthetic valve dysfunction , and most commonly .degenerative calcific aortic Valve. Echocardiographic evaluation of the severity of AR requires meticulous color-flow imaging, continuous-wave Doppler of the AR jet, and pulsedwave Doppler of the descending thoracic aorta and mitral inflow . Color-flow imaging of AR is best performed from the parasternal long- and short-axis views (by transthoracic echo window) or from the LVOT view (by window) . If there is no significant mitral regurgitation , mitral valve (MV) inflow can be used to represent systemic flow volume:

MV flow =MV annulus area x MV TVI

Where MV annulus area is determined by annulus diameter 2 x 0.785, and MV TVI by placing a sample volume at the MV annulus.

Regurgitant volume is the difference between stroke volume across the LVOT and mitral valve inflow, and regurgitant fraction (RF) is obtained by the following equation:

$$RF = \frac{\text{Regurgitant volume} \times 100}{\text{LVOT stroke volume}}$$

Based on the collective data from 2D Doppler and color-flow imaging, the severity of AR is determined as follow.

Severe AR is defined by

1. AR jet width / LVOT diameter ratio of 60% or more
2. AR jet area / LVOT area ratio of 60% or more
3. AR PHT less than 250 msec
4. Restrictive mitral flow pattern (in acute setting).
5. Holodiastolic flow reversal in the descending aorta
6. Dense continuous -wave Doppler signal
7. Regurgitation fraction of 55% or more
8. Regurgitation fraction of 60 mot or more
9. LV diastolic dimension of 7.5cm or more (chronic AR)

Mild AR is defined by

1. AR jet width / LVOT diameter ratio less than 30%
2. AR jet area / LVOT diameter ratio less than 30%
3. AR PHI more than 400 msec

4. Mild early diastolic flow reversal in the descending aorta

5. Faint continuous-wave Doppler signal

6. Regurgitant fraction less than 30%

7. LV diastolic dimension (chronic) of 6.0 cm or less

2 D/M-mode Echocardiograph

2 D/M-mode is useful in detecting the underlying etiology for mitral regurgitation such as mitral valve prolapse flail leaflet with or without ruptured chordae mitral annulus calcification, papillary muscle dysfunction or rupture, rhumatic valve, deft mitral valve, endocarditis, and perforation (mitral inflow) velocity may increase with severe regurgitation (especially with a prosthesis with a fixed orifice area) Regurgitant volume and fraction can be calculated to assess the severity of mitral regurgitation using 2D/Doppler studies [28] In the absence of significant AR, the difference between flow across the mitral valve and flow across the LVOT is mitral regurgitant volume, Flow across the mitral valve is calculated by the product of the mitral annulus area and the TVI of flow obtained by placing the sample volume at the mitral annulus. Flow across the LVOT is calculated by the product of the aortic annulus area and the TVI of flow obtained by placing the sample volume at the aortic annulus. Regurgitant fraction

is calculated by dividing regurgitant volume by flow across the mitral valve (MV) and multiplying by 100.

MV RV = flow - LOVT flow

$$\text{MV regurgitant fraction} = \frac{\text{MVRV}}{\text{MV flow}} \times 100\%$$

Where MV RN = MV regurgitant volume

Working Proforma

Name.....Age.....Sex.....Date.....

Echo No.....VCR Tape No.....

Height.....cms Weight.....kg BSA..... m^2....

Clinical Diagnosis

Quality of Imaging

Poor/Adequate/Good

MITRAL VALVE

Morphology : AML-Normal / Thickening / Calcification / Flutter / Vegetations / Prolapse / SAM /Doming

PML-Normal / Thickening / Calcification / Prolapse / Paradoxical Motion / Fixed

Subvalvular deformity - Present / Absent

Score.....

MV Area.....

Doppler: Normal / Abnormal

Mitral stenosis - present / Absent

EDG.....mmHG - MDG.....mmHg

MVA.....cm^2

Mitral regurgitation - Absent / Trivial / Mild / Moderate / Severe

TRICUSPID VALVE

Morphology:

Normal / Atresia / Thickening /Calicification / Prolapse / Vegetations / Doming

Doppler: Normal / Abnormal

Tricuspid Stenosis ; - Present / Absent

EDG.....mmHg - MDG.....mmHg

Tricuspid regurgitation - Absent/Trivial/Mild/Moderate/Severe

Velocity.....cm/sec. Pred RVS = PAP+RAP..... mmHg

PULMONARY VALVE

Morphology:

Normal /Atresia /Thickening /Doming /Vegetations

Doppler Normal/Abnormal

Pulmonary stenosis - Present/Absent Level

PSG.....mmHg

Pulmonary annulus.....mm

Pulmonary regurgitation - Present / Absent

Early diastolic gradient.....mmHg

End diastolic gradient.....mmHg

AORTIC VALVE

Morphology

Normal / Atresia / Thickening / Doming / Vegetations No. of Cusps 1/2/3/4

Doppler; Normal/Abnormal

Aortic Stenosis - Present/Absent Level

PSG.....mmHg

Aortic Annulus.....mm

Aortic regurgitation - Absent /Trivial /Mild /Moderate /Severe

MEASUREMENTS Normal Values Normal Values

Aorta.....(21-22 mm/m^2) LAed.....(21-22 mm/m^2)

LVes.....(21-40 mm) LVed.....(35-60 mm)

RVes.....(6-10 mm) PW (LV) ed-.....(7-11 mm)

RVed.....(20-38 mm) RV Anterior WAIL.....(upto 5 mm)

EF.....(62-80%)

IVS Motion Normal / Flat / Paradoxical

IAS

CHAMBERS

LV Normal /Enlarged /Clear /Thrombus /Hypertrophy

Contraction - Normal/Reduced

LA Normal /Enlarged /Clear /Thrombus

RA Normal /Enlarged /clear /Thrombus

RV Normal /Enlarged /Clear /Thrombus

PERICARDIUM Normal / Thickening / Calcification / Effusion

Wall Motion Analysis

Segmental Level	AS	ANT	ANT-LAT	POST-LAT	INF	IS
BASAL						
MID						
APICAL						

REMARKS

DIAGNOSIS

FINAL IMPRESSION

Observations

&

Results

Observation and Results

60 patients of chronic renal failure diagnosed by clinical biochemical and radiological parameters after using the inclusion criteria as described in material and methods were taken up for the study.

Detailed history and clinical examination were done prior to beginning of this study, routine investigations such as hemogram, blood urea, S. creatinine, USG abdomen etc were also done.

Echocardiographic examination was done in M.L.B. Medical College, Jhansi in the Department of Medicine, using a commercially available echocardiograph (Hewlett Packard 2000 CFM) interfaced with 2 - 2.5 Mhz dual frequency transducer.

The observations recorded are as under:

Demographic profile

Table 1

Age and Sex distribution

Age (years)	Males	Females
0 – 10	3 (5%)	Nil
10 – 20	1 (1.6%)	Nil
20 – 30	4 (6.6%)	2 (3.3%)
30 – 40	9 (15%)	5 (8.33%)
40 – 50	14 (23.1%)	7 (11%)
50 – 55	9 (15%)	6 (10%)
Total	40 (66.7%)	20 (33.3%)

Demographic characteristics

Age of study population was in the range of 8 – 55 years. Among 60 patients, 40 were males and 20 were females, males constituting 66% of study population and females constituting 34% of the study population. Male to female ratio was 2: 1.

Age and sex distribution

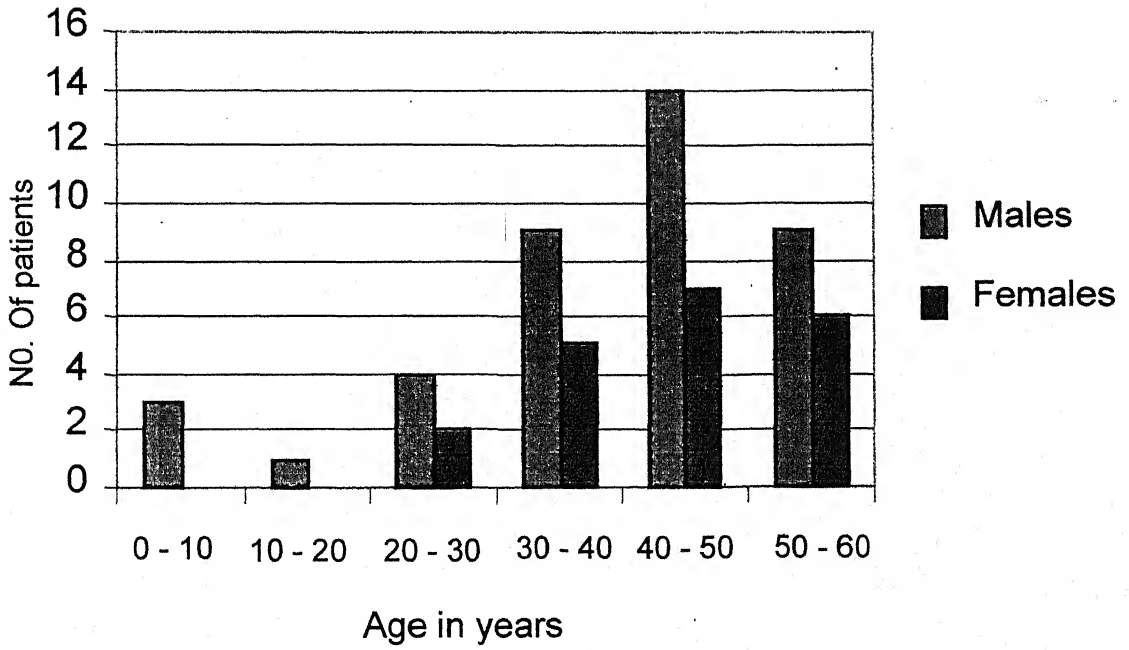


Table 2

Modality of treatment

Modality	Number of cases	Percentage (%)
Conventional treatment	18	30%
Hemodialysis	40	67%
Peritoneal dialysis	2	3%

Modality of treatment of patients were as follows : patients on conventional treatment were 18 (30%), on hemodialysis 40 (67%) and on peritoneal dialysis 2 (3%).

Table 3

Clinical & Biochemical profiles of 60 patients in three groups (A,B,C).

		Mild CRF	Moderate CRF	Severe CRF
		A	B	C
Serum Creatinine		1.5–3 mg/dl	3 – 6 mg/dl	> 6 mg / dl
Age (years)	Range	8 – 55	9 – 55	19 – 55
	Mean	40.5	42.3	50.1
Sex	Male	12	14	14
	Female	8	6	6
Biochemical parameters				
Blood urea (mg /dl)		40 ± 7.3	80 ± 16.3	136.5 ± 30.2
Serum Creatinine (mg /dl)		2.1 ± 0.5	3.9 ± 0.9	9.8 ± 3.6
Hb (g / dl)		13.2 ± 1.5	9.3 ± 1.8	7.3 ± 1.7

	Mean	S.D.	Number of patients
Blood Sugar	113.77	± 7.62	60
Sodium	137.2	± 5.97	60
Potassium	4.1	± 0.63	60

The patients were randomized in 3 groups, A, B and C (Mild , Moderate and Severe chronic renal failure) according to serum creatinine, clinical and biochemical profile of the patients as given in the table.

Clinical examination and USG abdomen examination

The mild renal failure patients were having serum creatinine in the range of 1.5 – 3.0 mg/dl, moderate renal failure patients having serum creatinine in the range of 3.0 – 6.0 mg/dl and those with severe renal failure having serum creatinine in the range of > 6 mg / dl.

Table 4

Echocardiographic parameters for left ventricular mass index in three groups (A, B, C).

	Mild CRF	Moderate CRF	Severe CRF
	A	B	C
LVID (cm)	3.4 ± 0.3	3.9 ± 0.6	4.7 ± 0.5
IVS (cm)	1.2 ± 0.1	1.5 ± 0.3	1.7 ± 0.3
PWD (cm)	1.0 ± 0.1	1.2 ± 0.2	1.3 ± 0.2
LVMI	73.9 ± 14.8	122.1 ± 34.7	175.2 ± 36.4

Table 5

Left Ventricular Mass Index and its Sex wise distribution in three groups (gm/m^2)

		Mild CRF	Moderate CRF	Severe CRF
Whole group		A	B	C
	Mean	73.8	122.1	175.2
	S.D.	14.8	34.7	36.4
Male	Mean	76.5	122.02	183
	S.D.	15.6	14.5	37.9
Female	Mean	71.1	121.4	164.9
	S.D.	15.9	50.9	31.9

ECG findings in CRF patients

	Mild CRF	Moderate CRF	Severe CRF
	A	B	C
Total no. of patients	20	20	20
No. of patients having LVH in ECG	4	4	10

ECG findings in chronic renal failure patients based on Romhilt Estes criteria for diagnosis of LVH, 4 patients with mild chronic renal failure, 4 patients with moderate chronic renal failure and 10 patients with severe chronic renal failure were having LVH. Out of 60 patients of chronic renal failure, 18 (30%) were having positive ECG findings for LVH.

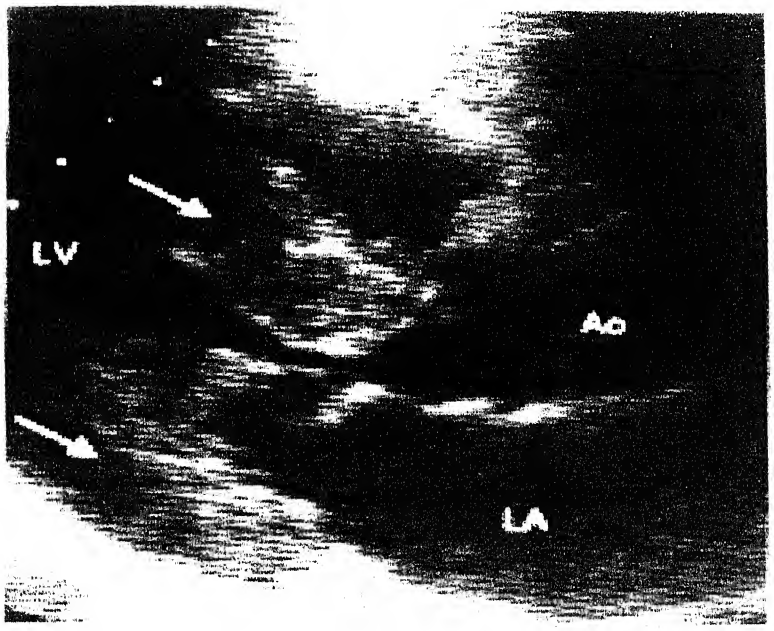
Table 6

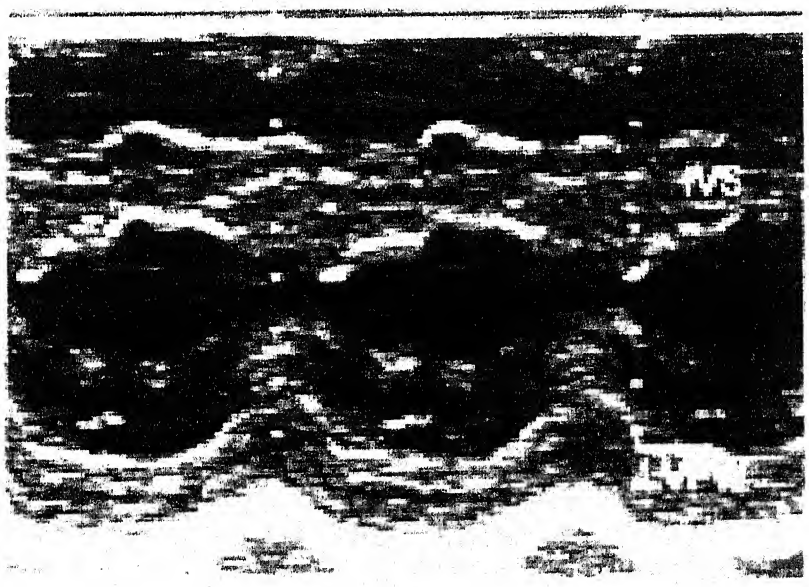
	Mild CRF	Moderate CRF	Severe CRF
	A	B	C
Total number of males	12	14	14
Number of males with LVH	2 (16%)	3 (23.5%)	13 (94.1%)
Total number of females	8	6	6
Number of females with LVH	Nil	4 (66%)	6 (100%)

Left Ventricular Hypertrophy

Total number of males found to be having LVH was 28 out of 60 i.e. 47.6% out of which 18 (64%) were males and 10 (35.7%) were females, among which 2 patients (16%) out of 20 patients in group A were having LVH and 7 patients (3 males (23.5%) and 4 females (66%)) were having LVH in group B, i.e. moderate chronic renal failure.

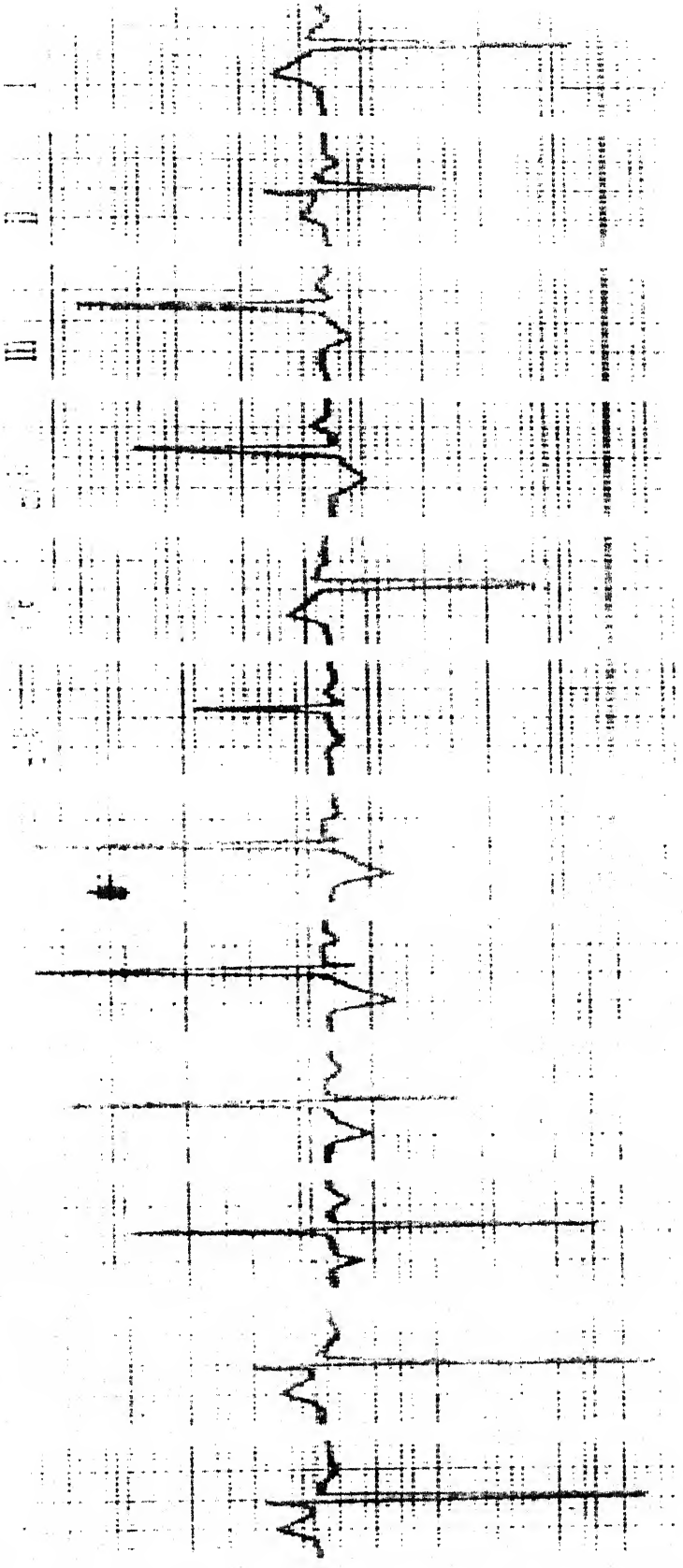
Nineteen patients in group C, i.e. severe chronic renal failure, were found to be having LVH out of which 13 (94.1%) out of 14, were males and 6 out of 6 patients (100%) were females.





L.V.H

ECG showing L.V.H



LVH in three groups

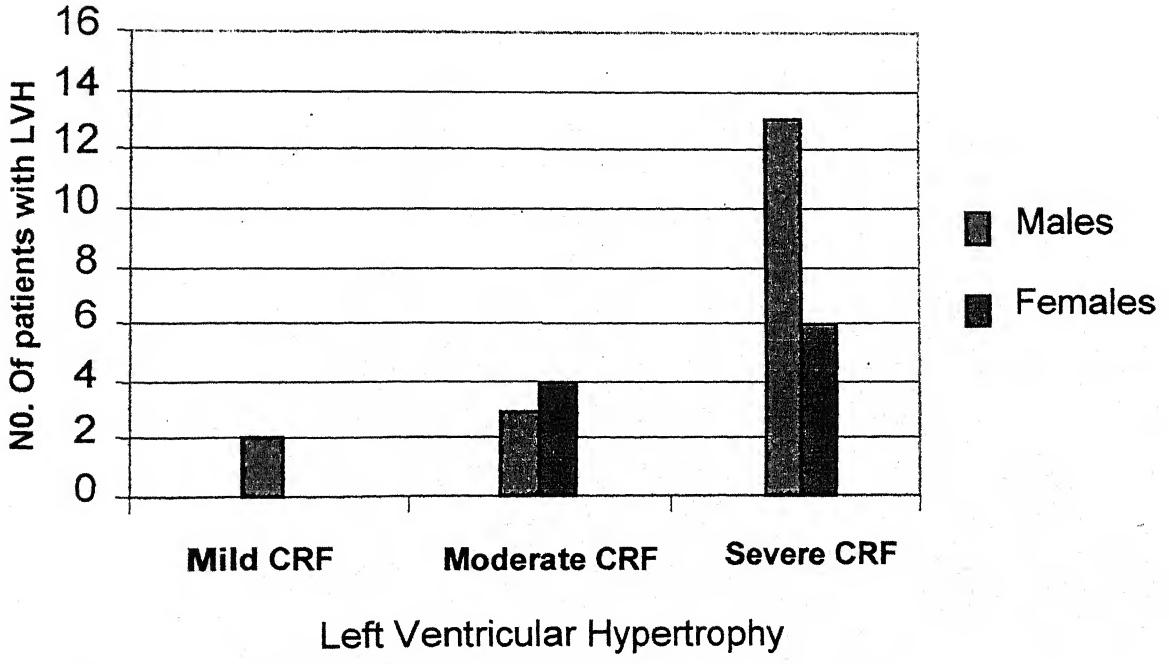
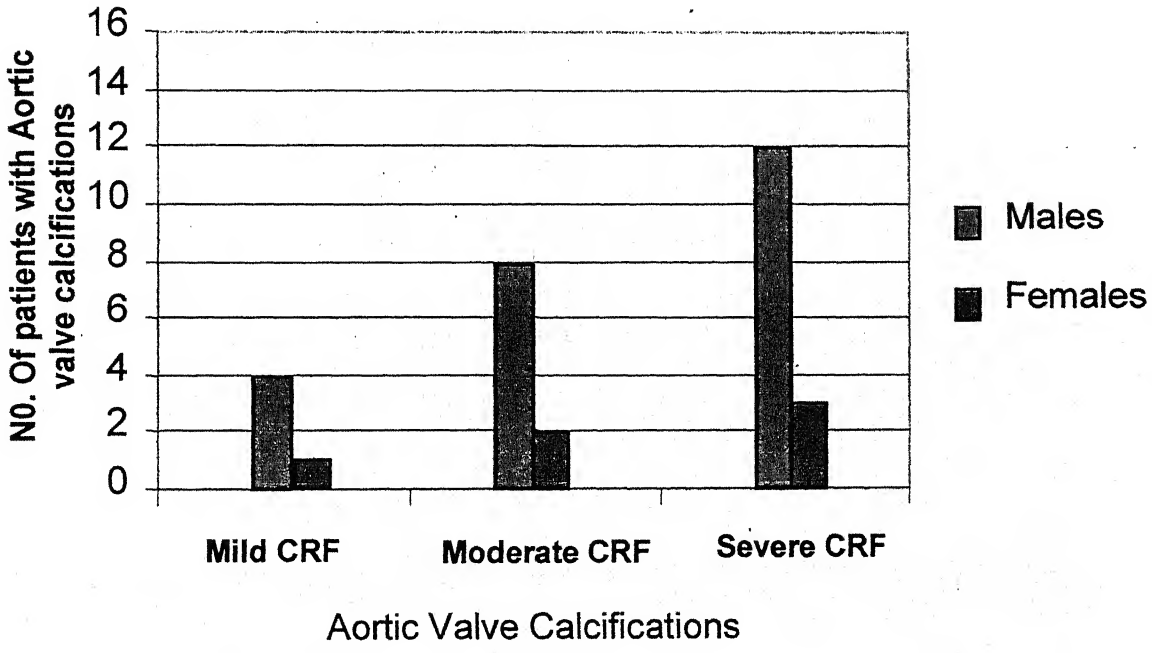


Table 7

Cardiac calcifications

	Mild CRF	Moderate CRF	Severe CRF
	A	B	C
Total number of males	12	14	14
Number of males having calcification over different valves			
Aortic valve	4 (33%)	8 (54%)	12 (84%)
Mitral valve	Nil	Nil	Nil
Tricuspid valve	Nil	Nil	Nil
Pulmonary valve	Nil	Nil	Nil
Total number of females	8	6	6
Number of females having calcification over different valves			
Aortic valve	1 (16%)	2 (33.3%)	3 (50%)
Mitral valve	Nil	Nil	Nil
Tricuspid valve	Nil	Nil	Nil
Pulmonary valve	Nil	Nil	Nil

Aortic valve calcification in three groups



Valvular calcification

Out of 60 patients, 30 patients (50%) were found to be having aortic valve calcification. Out of 30 patients having aortic valve calcification, 24 were males and 6 were females, and it was seen that aortic valve calcification was more in severe chronic renal failure patients.

In group A (mild chronic renal failure) 4 males (33%) out of 12 males, in group B (moderate chronic renal failure) 8 males (54%) out of 14 males and in group C (severe chronic renal failure) 12 (84%) males out of 14 males were having aortic valve calcification.

In female population in group A (mild chronic renal failure) 1 female (16%) out of 8 females, in group B (moderate chronic renal failure) 2 females (33.3%) out of 6 females and in group C (severe chronic renal failure) 3 females (50%) out of 6 females were having aortic valve calcification. In this way aortic valve calcification was more in severe chronic renal failure group and in older age. Calcification on other valves were absent.

Table 8

Pericardial effusion

	Mild CRF	Moderate CRF	Severe CRF
	A	B	C
Total number of patients	20	20	20
Total number of males	12	14	14
Number of males with pericardial effusion	Nil	1 (7%)	2 (14%)
Total number of females	8	6	6
Number of females with pericardial effusion	Nil	Nil	1 (16%)

Pericardial Effusion

The same three groups were also studied for pericardial effusion. Pericardial effusion was found in 4 (6%) of patients out of total 60 patients. Out of 4 1 patient (25%) was female and 3 patients (75%) were males.

Pericardial effusion in three groups

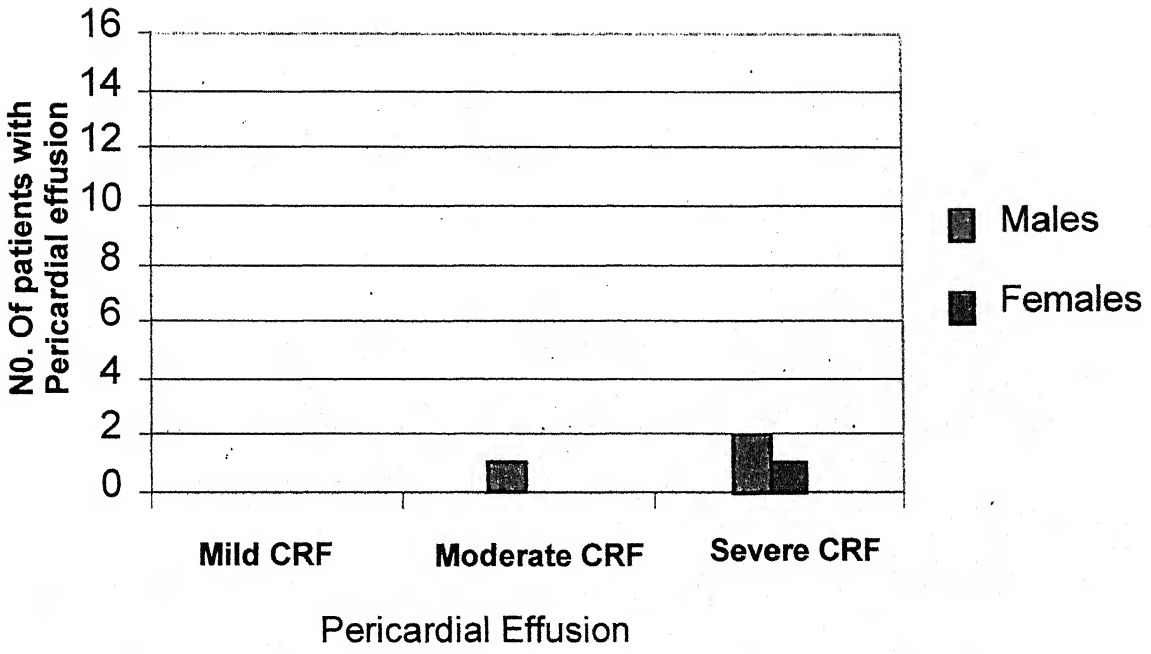


Table 9

Congestive Heart Failure

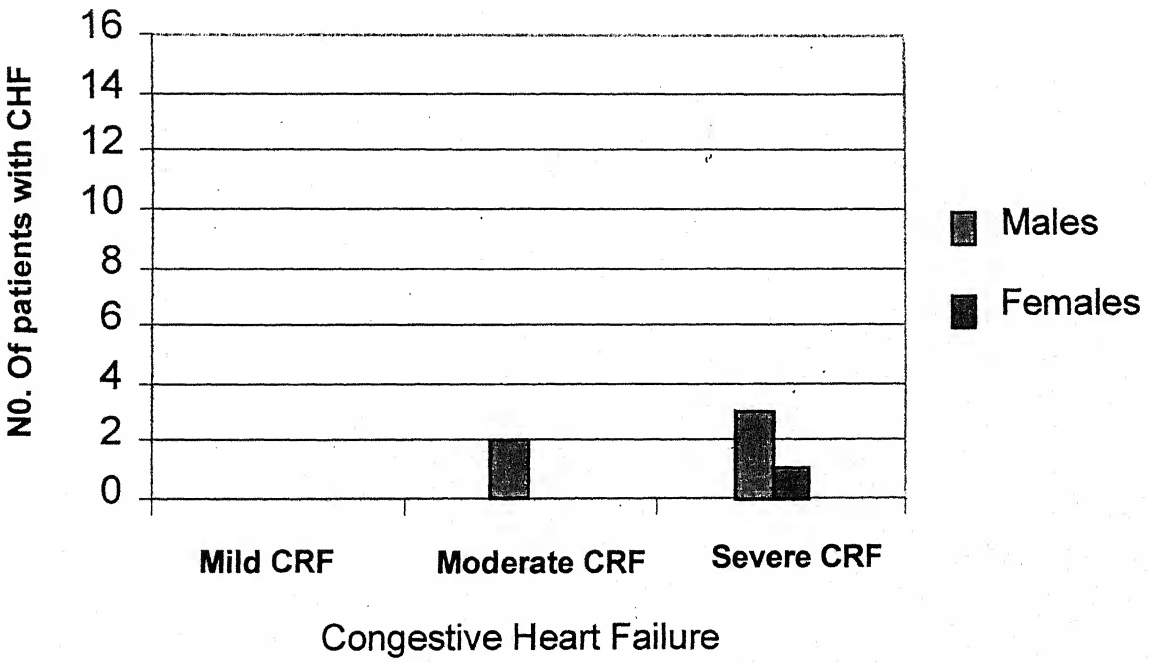
	Mild CRF	Moderate CRF	Severe CRF
	A	B	C
Total number of patients	20	20	20
Total number of males	12	14	14
Number of males with CHF	Nil	2 (14%)	3 (21.1%)
Total number of females	8	6	6
Number of females with CHF	Nil	Nil	1 (16.6%)

Patients having CHF with moderate chronic renal failure, ejection fraction was 42 ± 3% patients with severe chronic renal failure having ejection fraction 35 ± 2% and in patients with mild and normal chronic renal failure having an ejection fraction of 60 ± 3%.

Chronic Heart failure

Same groups were also studied for chronic heart failure and 6 patients (10%) out of 60 patients were found to be having chronic heart failure, and they were having systolic dysfunction.

Congestive Heart Failure in three groups



Patients having chronic heart failure with chronic renal failure were having an ejection fraction of 42% ± 3% and patients having severe chronic renal failure with chronic heart failure having an ejection fraction of 35% ± 2%

Chronic heart failure was found to be more in severe chronic renal failure and infective endocarditis, D.C.M were nil in these patients.

Discussion

Discussion

Premature cardiovascular disease is a significant cause of morbidity and mortality among the patients with CRF.

Four main structural abnormalities of heart have been described in patients with CRF i.e LVH, myocardial calcification, pericardial effusion and CHF.

All these abnormalities promote systolic as well as diastolic dysfunction which predisposes to symptomatic heart failure, which is a risk factor for premature death. Various diagnostic modalities, both invasive and non-invasive such as ECG, echocardiography and radionucleide scans are utilized for diagnosing LVH and left ventricular dysfunction.

Echocardiography provides an excellent non-invasive method to delineate details of anatomy of cardiac cavity, wall dimensions and wall movements. It is now increasingly used in the assessment of cardiac performance and is also invaluable in the demonstration of structural abnormalities such as LVH, pericardial effusion, CHF and cardiac calcification.

In the present study out of 60 patients 28 (47%) were found to be having LVH. These results confirm to prevalence of LVH in patients with ESRD from 40% to 80% in various studies. Raj et al, (1997) found prevalence of LVH in undialysed CRF patients as 45.7% and in patients requiring dialysis as 76.5%, which is similar to our study.

Levin et al, (1996) found prevalence of LVH in predialysis population to be 38.9%. They also demonstrated that prevalence of LVH increases with the progressive decline in renal functions.

To conclude the present study shows that the patients with chronic renal failure have higher left ventricular mass index and higher prevalence of LVH, which is more marked in patients with severe chronic renal failure.

The prevalence of LVH even in predialysis patients on echocardiography implies that these patients require detailed cardiovascular evaluation despite absence of symptoms and also that various efforts aimed at prevention and control of LVH should be started early during the course of renal insufficiency.

ECG findings in chronic renal failure patients based on Romhilt Estes criteria for diagnosis of LVH – 4 patients with mild chronic renal

failure, 4 patients with moderate chronic renal failure and 10 patients with severe chronic renal failure were having LVH. Out of 60 patients of chronic renal failure, 18 (30%) patients were having positive ECG findings for LVH.

In the present study **aortic valve calcification** was found in 30 (50%) patients out of 60. Our findings are in almost similar to the results of Raine (1994), who showed a high prevalence of aortic valve calcification (55%) on 2D echocardiography.

Aortic valve calcification was found more in older age group.

The same number of patients were subjected for evaluation of pericardial effusion. Four (6%) patients out of 60, were found to be having pericardial effusion. Out of which 1 patient was female and 3 were males. The pericardial effusion was more in advanced chronic renal failure.

All 60 patients were considered for CHF evaluation, the patients having CHF with moderate chronic renal failure were having an ejection fraction of 42 ± 3% and in patients having severe chronic renal failure the ejection fraction was 35 ± 2%. Normal patients as well as patients with mild chronic renal failure were having an ejection fraction of 60 ± 3%.

Total number of patients having CHF were 6 (10%) out of total 60 patients. All the 6 patients were found to be having systolic dysfunction. CHF was more in advanced renal failure.

Infective endocarditis, dilated cardiomyopathy and mitral valve calcification was absent in all the 60 patients.

Conclusion

Conclusion

To conclude the present study shows that the patients with CRF have higher prevalence of LVH (47%), CHF (10%), pericardial effusion (6%), cardiac calcification such as aortic valve calcification (50%) and all of them were found to be more in severe chronic renal failure patients.

Thus, it has been observed that CRF patients are prone for development of various cardiac abnormalities such as LVH, pericardial effusion, cardiac calcification, and CHF.

Cardiovascular evaluation is necessary for prevention and control of various cardiac abnormalities and prevention of mortality and morbidity from cardiac causes.

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